As an obiter dictum, I request the inclusion of caliber and muscle velocity as a requisite for publication. The excuse that caliber cannot be determined is not persuasive. Caliber information can often be obtained from the police (type of weapon, spent cartridges, etc.) and from simple bullet measurements derived from the x-ray (2).

Jules C. Ladenheim
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1. Heary RF, Vaccaro AR, Mesa JJ, Northrup BE, Albert TJ, Balderston RA, Cotler JM: Steroids and gunshot wounds to the spine. **Neurosurgery** 41:576–585, 1997.
2. Ladenheim JC, Ladenheim ED: *Firearms and Ballistics: For Physician and Attorney.* Bogota, Statia Publ., 1996, pp 277, 299.

In reply: We agree with Ladenheim that information regarding the muzzle velocity and bullet caliber would be valuable. This problem highlights the differences that occur between experimental studies and clinical research efforts. In the article by Ladenheim and Ladenheim (1), differences in velocity were clearly shown to cause different injury patterns. In the clinical study we presented, the actual bullet and weapon were rarely recovered, which meant that any comments would be pure conjecture. As Ladenheim points out in his textbook, bullet measurements derived from x-rays may be useful. Including measurement data in our article would have represented fewer than 20% of the total cases. Because these bullets all caused spine injuries, many of them were appreciably deformed by their impact with the bony spine, which would render any prediction regarding the bullet caliber the subject of speculation.

Ladenheim has postulated that low velocity injuries might respond to steroids. This was not the case with our patients. More than 97% of the injuries in this study were caused by low-velocity weapons; however, steroids did not prove to be of any benefit in these patients.

We appreciate Ladenheim's comments concerning our article. We did not have adequate data regarding muzzle velocity and bullet caliber to be able to perform the correlation that he seeks.

Robert F. Heary
Alexander R. Vaccaro
Newark, New Jersey

1. Ladenheim JC, Ladenheim ED: *Firearm and Ballistics: For Physician and Attorney.* Bogota, Statia Publ., 1996.

Intramedullary Pressure in Syringomyelia: Clinical and Pathophysiological Correlates of Syrinx Distension

To the Editor: The article by Milhorat et al. (2) claims to be "the first to measure intramedullary pressure in a human disease" in syringomyelia. Unfortunately, both the authors and the four distinguished commentators seem to be ignorant of the fact that syrinx pressures have been measured for more than 15 years.

Seventeen cases were reported in 1989 (1), and the fascinating and inexplicable "inherent collapsing pressure" has been described in detail. The question of pressure measurement in relation to intracyst valve shunting has been extensively described (3).

Charles H.G. Davis
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1. Davis C, Symon L: Mechanisms and treatment in post-traumatic syringomyelia. **Br J Neurosurg** 3:669–674, 1989.
2. Milhorat TH, Capocelli AL Jr, Kotzen RM, Bolognese P, Heger IM, Cottrell JE: Intramedullary pressure in syringomyelia: Clinical and pathophysiological correlates of syrinx distension. **Neurosurgery** 41:1102–1110, 1997.
3. Suzuki M, Davis C, Symon L, Gentili F: Syringoperitoneal shunt for treatment of cord cavitation. **J Neurol Neurosurg Psychiatry** 48:620–627, 1985.

In reply: Davis points out that in our recently published article, we failed to cite his earlier work on syrinx pressures. I offer my sincerest apologies. The findings to which Davis refers were not heralded by the title of the article (1), and the article lacked a key words list. The work thereby eluded a diligent and exhaustive computerized search of the literature. I have subsequently read the article and commend it. I will cite it in future references to the subject.

Thomas H. Milhorat
Brooklyn, New York

1. Davis C, Symon L: Mechanisms and treatment in post-traumatic syringomyelia. **Br J Neurosurg** 3:669–674, 1989.

Complications in Patients with Palmar Hyperhidrosis Treated with Transthoracic Endoscopic Sympathectomy

To the Editor: Hyperhidrosis of the palms is a common problem and a classical indication for transthoracic endoscopic sympathectomy. However, compensatory sweating occurring in the majority of patients (1, 3) may impede patients' satisfaction with the outcome of the procedure (3). We achieved a successful outcome with the application of botulinum-toxin A (BT-A) to abolish this side effect in a severely affected patient.

A 48-year-old Caucasian man who had suffered from severe palmar hyperhidrosis since puberty had undergone numerous therapeutic attempts elsewhere, including transthoracic endoscopic sympathectomy that eventually resolved the hyperhidrosis of his palms but resulted in extensive compensatory perspiration in a circumscribed area of approximately 30 × 40 cm on the right side of his trunk. Subsequently, he had to endure sweat-soaked clothes over the affected area despite his complete redressing up to six times per day. He developed rheumatic pain because of this half-sided reduction in skin temperature in the hyperhidrotic region.

After providing written informed consent, the patient received an intradermal administration of BT-A, which is a potent blocker of cholinergic synaptic transmission, as described by Tsui (3). A total of 400 units of BT-A (Dysport; Speywood Pharmaceuticals, England) diluted in 2 ml of normal saline was injected strictly intradermally in 0.1-ml aliquots spread evenly over one-half of the area of the hyperhydrotic region in

one session. After 2 days, the treated area was completely anhidrotic whereas the surrounding region remained hyperhydrotic, as reported by the patient and confirmed by an iodine-starch test and by quantification of sweat secretion by weighing standardized blotting sheets (gravimetry). The skin temperature was 36.9°C in the treated region and 33.6°C in the immediately neighboring region, as determined by infrared sensory point thermometer (OPTRON, Neuenkirchen, Germany). Because no muscular weakness or any systemic side effects occurred, the remaining hyperhidrotic region was treated accordingly, relieving the patient completely from his hyperhidrotic symptoms, including his related rheumatic complaints (14 mo after treatment), without the occurrence of compensatory sweating in other areas of the body.

Because acetylcholine transmits sympathetic activity exclusively in the cutaneous sweat gland, it can be specifically blocked by intradermal injections of BT-A. This may offer an effective therapeutic modality for selected patients suffering from recalcitrant compensatory sweating. Further investigation is needed to determine whether this procedure may result in pertinent normalization of sympathetic outflow to the skin.

Marc Heckmann
München, Germany

1. Byrne J, Walsh TN, Hederman WP: Endoscopic transthoracic electrocautery of the sympathetic chain for palmar and axillary hyperhidrosis. **Br J Surg** 77:1046–1049, 1990.
2. Lai YT, Yang LH, Chio CC, Chen HH: Complications in patients with palmar hyperhidrosis treated with transthoracic endoscopic sympathectomy. **Neurosurgery** 41:110–115, 1997.
3. Tsui JKC: Botulinum toxin as a therapeutic agent. **Pharmacol Ther** 72:13–24, 1996.

In reply: Compensatory hyperhidrosis (CH) is the most annoying side effect of surgical treatment for palmar hyperhidrosis (PH). I have surgically treated more than 200 cases of palmar hyperhidrosis using open or endoscopic approaches. In these patients, more than 90% experienced postoperative CH. I think that, at least in Taiwan, CH is the price we must pay for the relief of PH. In our study, the surgical approaches (open or endoscopic sympathectomy) and the extent of sympathectomy (T2 only or T2 plus T3) made no difference in the incidence and severity of postoperative CH.

Modified or limited ganglionectomy (partial destruction of the main ganglion at T2) has been advocated by some surgeons in Taiwan to prevent CH. We think that this is either ineffective or prone to surgical failure. Limited ganglionectomy for PH carries the potential risks of surgical failure and recurrence. Recently, we experienced a recurrent case in a patient who had undergone so-called "modified" ganglionectomy at another hospital 2 years previously. He experienced temporary relief of PH with the side effect of significant CH after undergoing partial ganglionectomy. One and a one-half years later, his PH recurred and he also noted the progressive subsidence of CH! We reoperated, performing thoracoscopic surgery, and an indistinct pleural scar over a "completely regenerated" T2 ganglion, indicating the previous operation, was observed. We performed a bilateral T2 sympathectomy, as routine, and achieved successful relief of the recurrent PH but incurred the inevitable side effect of CH.

CH is the main cause of patients' dissatisfaction after surgery for relief of PH. We are happy to know that Heckmann achieved a successful outcome using the intradermal administration of BT-A over the hyperhydrotic area to treat a case of CH. We wonder, for cases of CH in which the regions are widely distributed over chest, back, and both thighs, whether large dosages of BT-A are needed for intradermal injection. Would this be practical? Would any adverse effects be associated with the large dosages?

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